

Durham Research Online

Deposited in DRO:

12 May 2014

Version of attached file:

Accepted Version

Peer-review status of attached file:

Peer-reviewed

Citation for published item:

Schrecker, Ted and Acosta, Lisa and Somerville, Margaret A. and Bursztajn, Harold (2001) 'The ethics of social risk reduction in the era of the biological brain.', *Social science medicine*, 52 (11). pp. 1677-1687.

Further information on publisher's website:

[http://dx.doi.org/10.1016/S0277-9536\(00\)00281-1](http://dx.doi.org/10.1016/S0277-9536(00)00281-1)

Publisher's copyright statement:

This is the author's version of a work that was accepted for publication in *Social Science Medicine*. Changes resulting from the publishing process, such as peer review, editing, corrections, structural formatting, and other quality control mechanisms may not be reflected in this document. Changes may have been made to this work since it was submitted for publication. A definitive version was subsequently published in *Social Science Medicine*, 52, 11, 2001, 10.1016/S0277-9536(00)00281-1.

Additional information:

Use policy

The full-text may be used and/or reproduced, and given to third parties in any format or medium, without prior permission or charge, for personal research or study, educational, or not-for-profit purposes provided that:

- a full bibliographic reference is made to the original source
- a [link](#) is made to the metadata record in DRO
- the full-text is not changed in any way

The full-text must not be sold in any format or medium without the formal permission of the copyright holders.

Please consult the [full DRO policy](#) for further details.

The Ethics of Social Risk Reduction

Ted Schrecker, M.A.*
176 York Street, Suite 31
London, Ontario N6A 1B5 CANADA
Phone: 519 871 3630 Facsimile: 519 432 5649 E-mail: tschrecker@sympatico.ca

Lisa Acosta, B.A.
Rush Medical College
1524 West Harrison, #2A
Chicago, Illinois 60607 USA
Phone: 312 733 8851 E-mail: LAcosta@rushu.rush.edu

Margaret A. Somerville, D.C.L., A.M., FRSC
Gale Professor of Law, McGill Centre for Medicine, Ethics and Law
3690 Peel Street
Montréal, Québec H3A 1W9 CANADA
Phone: 514 398 7401 Facsimile: 514 398 4668 E-mail: somerv_m@falaw.lan.mcgill.ca

Harold J. Bursztajn, M.D.
Associate Clinical Professor of Psychiatry and Co-director, Program in Psychiatry and Law
Harvard Medical School, Cambridge Massachusetts 02138 USA Phone: 617 492 8366
Facsimile: 617 441 3195 E-mail: harold_bursztajn@hms.harvard.edu

* corresponding author; address as of 15 July 2000: 450, rue de la Congrégation, Montréal, Québec H3K 2H7 CANADA. Phone and facsimile numbers not yet known; e-mail will remain the same.

The Ethics of Social Risk Reduction in the Era of the Biological Brain

Abstract

In keeping with our transdisciplinary orientation, in this article we try to do several things at once. We address research on preventing mental illness and its relation to existing conceptions of public health, a topic to which insufficient attention has been paid in the era of the biological brain, while using this case study to illustrate the limits of conventional approaches in bioethics. After identifying the crucial need for methodological self-consciousness in prevention research and policy, we explore the implications as they relate to (i) the values embedded in the choice of research designs and strategies, and (ii) contrasting intellectual starting points regarding the biological plausibility of preventing mental illness. We then draw attention to the need for more thoughtful analysis of the appropriate role and limits of economics in making choices about prevention of mental illness.

Keywords

Mental illness; prevention; public health; methodology; economics; ethics.

The Ethics of Social Risk Reduction in the Era of the Biological Brain

Dedication

This article is dedicated, with deep respect and a sense of great loss, to the late Jonathan Mann. Among the many ways he contributed to making the world a better place, his work in joining ethical analysis and public health, both in theory and in practice, has given to us all a legacy for the future. For this and much more, we are all in his debt.

I. Introduction

The chapter on prevention in a recent text on mental health planning begins: “The proposition that it is better to prevent illness than to allow it to occur with its associated suffering, loss of function, and need for treatment, is one of the basic premises of public health that requires no justification” (Breakey, 1996, p. 323). Public health as a profession and a field of inquiry is undeniably organized around the premise that prevention is valuable; thus, the substance of public health has been defined as “organized community efforts aimed at the prevention of disease and the promotion of health” (Remington et al., 1988, p. 41). Attaching high priority to prevention also fits well with the commitments to non-maleficence and beneficence that are widely acknowledged as

central principles of bioethics (Beauchamp and Childress, 1994, pp. 189-325). However, few propositions in public health or public policy require no justification. This point is particularly evident in the literature on preventing mental illness, where the case for preventive interventions is often made primarily on economic grounds. Implicit in this argument, if seldom acknowledged or defended, is the premise that allocations of resources to prevention require justification, and some don't make the grade.

This is a normative proposition; it can be neither established nor refuted on scientific grounds. In this respect as in several others, the prevention of mental illness raises complex ethical issues at the interface of science, public policy, law and clinical practice. Such issues have not been fully addressed in contemporary bioethics, because of the field's preoccupation with two particular frames: the clinical frame, in which ethical analysis is organized around physician-patient relationships, and the human subjects research frame, in which analysis is organized around researcher-subject relationships. In each case, the focus is on relations and transactions among individuals. In this article we explore several clusters of issues surrounding prevention, public health and mental illness, focusing not on individuals but rather on how the biological and biomedical approaches that dominate research on mental illness have distracted attention from public health as the project of "assuring the conditions in which people can be healthy" (Remington et al., 1988, p. 40; Mann, 1997, p. 7). Our approach insists that ethical analysis must be expanded to include issues and choices that arise in the choice of research methodologies and strategies themselves. Although critiques of the ideal of value-free research are nothing new, the implications of such critiques as applied to research on the etiology and prevention of mental illness have not been adequately explored.

Central to this argument is the importance of what Shrader-Frechette and McCoy (1993, pp. 84-101), drawing on the work of Helen Longino (1990), have called methodological value judgments. This concept reflects the earlier insight of Thomas Kuhn (1970, pp. 199-200) concerning the impossibility of any “neutral algorithm for theory-choice.” It does not mean there is anything unscientific about such choices, and identifying them does not imply that the scientists who make them are doing bad science, or conducting themselves unethically. Indeed,

... scientists make methodological value judgments whenever they follow one methodological rule, rather than another. For example, whenever one uses a particular research design because of available computer software, one is making a methodological value judgment that the research design is adequate. Even collecting data requires use of methodological value judgments because one must make evaluative assumptions about what data to collect and what to ignore, how to interpret the data, and how to avoid erroneous interpretations. (Shrader-Frechette and McCoy, 1993, p. 84)

For example, a cross-sectional questionnaire survey used to study the relation between homelessness and serious mental illness will yield a different *kind* of result from that obtainable by way of a longitudinal ethnography, and those differences will persist even if each study is a paragon of methodological rigour on its own terms. Like the choice to make the case for preventive interventions primarily on economic grounds, the choice of methodologies is not exclusively a scientific one. Neither, however, is it merely a matter of opinion; the value judgments that enter the choice among possible research designs and strategies must reflect the same intellectual rigour as the actual conduct of research, whether conducted in the laboratory or in the field.

Central to our argument, as well, is the inadequacy of some conceptions of prevention when assessed against the demands of “ensuring the conditions in which people can be healthy”.

An important, and increasingly widespread set of methodological value judgments limits the universe of measures to achieve this goal, as it applies to mental illness, by circumscribing the range of research designs whose results are considered credible. Consequently, we suggest that discussions of public health and mental illness should refer not only to prevention, but also (and in some instances preferably) to social risk reduction. This position reflects an increasingly broad acceptance of the importance of social determinants of health (Evans and Stoddart, 1990), but it is controversial because it implies at least provisional acceptance of a connection between the social environment and mental illness that some would view as unproved, or even ideological (Heller, 1996, p. 1124). Conversely, the provisional rejection of that connection can likewise be regarded as ideological (Albee, 1996).

Our approach emphasizes the importance of transdisciplinarity and methodological pluralism, but goes further in terms of identifying the central importance of methodological self-consciousness. Certain background assumptions (Longino, 1990, pp. 59-61, 108-112, 117-121, 133-161) about how the world works are *unavoidably* embedded in the choice of research designs and strategies, and in other areas of the public health enterprise. These assumptions may have significant ethical implications when they are embodied in research leading to findings that are subsequently regarded as authoritative, whether in clinical practice or in public policy. Our position also implies the need for engagement with macro-level debates about at least some aspects of social policy, particularly with respect to such initial presumptions (Somerville, 1989, p. 539; Somerville, 1993, pp. 64-68) as those having to do with the appropriate role of economic analysis in public health. Since interventions aimed at reducing the incidence or the destructive consequences of mental illness will probably involve substantial allocations of societal

resources, as they have historically done in other areas of public health, a direct encounter between ethics and public policy is unavoidable, and indeed should be welcomed.

II. Prevention Research: Values in Research Design

Recent research on preventing mental illness reflects the drift in contemporary epidemiology away from determinants of population health, and toward a focus on individuals within a high-risk population. Lost in that shift are important questions about why *populations* differ in health status (Krieger and Zierler, 1996; McMichael, 1995; Pearce, 1996), and about the connections between those differences and the social determinants of health. The shift reflects an important methodological value judgment, and underscores the importance of our earlier discussion of this topic. In terms of the conclusions that follow for purposes of trying to prevent mental illness, there is an important difference between asking why certain individuals in a high-risk population suffer from a particular disorder and asking why the incidence of the disorder is much higher in particular populations.

A particularly dramatic contrast emerges from two lines of research on PTSD. Whereas research on PTSD among prostitutes documents an incidence that is higher than some studies have found among combat veterans (Farley and Barkan, 1998; Farley et al., 1998), a recent article and an accompanying editorial in the *Canadian Journal of Psychiatry* (Bowman, 1999; Paris, 1999) ask instead why some people appear relatively invulnerable to PTSD, and emphasize the potential significance of hereditary differences in susceptibility. As in research on other potentially disabling mental disorders, it is important to ask both why so many members of a particular population develop the disorder and why some do not. Each set of questions is important, but the

choice to organize research primarily or exclusively around one or the other embodies a methodological value judgment of some importance. The need to scrutinize such choices with specific attention to their ethical and policy implications, and to the initial presumptions they embody, is not adequately emphasized in existing approaches to ethics in public health research.¹

As another example, Muñoz et al. (1995) reported qualified success in a randomized controlled trial of “a course on cognitive behavioural methods to gain greater control of one’s mood” as an initiative to prevent major depression among members of a high-risk population -- patients of San Francisco outpatient primary care clinics serving low-income and minority populations. Along almost every dimension (class, race, gender, relatively advanced age, relatively limited education, disability) the demographics of the sample reflected the fault lines that define economic vulnerability and marginalization in contemporary North America.² Now, it is obviously worthwhile to know about the effectiveness of initiatives like the one studied in San Francisco, which are defined with reference to the responsibilities and budgets of mental health professionals who must work within a particular institutional framework. An alternative research perspective, on a different scale, might ask such questions as: were vulnerability and marginalization depressogenic at the level of the individual subjects? How might the life events associated with low income and economic insecurity be involved? The answers to this set of

1. For an illustration of this omission, see Coughlin and Beauchamp, eds., 1996.

2. Just over one-third (35.1%) were white, with African Americans (23.7%) and Latinos (24.3%) comprising the next two largest ethnic/racial groups. “Their mean age was 52.5 years, mean income \$11,500, and mean years of education 12.1. Their unemployment rate was very high (67.8%).” Thirty-nine of the unemployed subjects, or 26 percent of the entire sample, were unemployed by reason of disability, and another 21 (14 percent of the entire sample) were looking for work. Ninety-three of the 150 participants, or almost two-thirds, were female. (Muñoz et al., 1995, pp. 202-205)

questions might in turn suggest quite different, albeit more ambitious and longer term, directions for preventive intervention – or, to use our preferred terminology, social risk reduction.

Several points need to be made about this example before proceeding any further. No criticism is intended of the San Francisco project researchers, who were acutely aware both of the special vulnerabilities associated with their study population and of the broader background conditions against which their study took place. Depression may be a special case, since the social and economic risk factors are probably better established than in the case of other major mental illnesses (Mrazek and Haggerty, eds., 1994, pp. 165-171). Nevertheless, such factors as poverty and social disorganization may be relevant across the spectrum of severe mental illness, especially in terms of opportunities for what would conventionally be described as secondary or tertiary prevention (Cohen, 1993; Hiday, 1997; Ware and Goldfinger, 1997). Some of the questions asked in the preceding paragraph can only be answered by way of qualitative methods, preferably undertaken longitudinally. (The underutilization of qualitative methods in mental illness research deserves discussion in a separate article.) Finally, the example suggests a contrast between what might be called *clinical* and *contextual* foci for prevention research. The contrast is generically important, and one can find numerous other illustrations.

For instance, low birth weight has been identified as a general risk factor for a variety of adverse health outcomes, including those related to mental illness (Mrazek and Haggerty, eds., 1994, pp. 182-183). Improving the adequacy of prenatal care appears to be important in reducing low birth weight, and “lack of prenatal care has important implications for mental disorders” (Mrazek and Haggerty, eds., 1994, pp. 222-225). At least in the United States, inadequate prenatal care is directly related to economic vulnerability, particularly when combined with residence in “geographic pockets of need” (Brown, ed., 1988, pp. 26-53). Financial factors such as lack of

insurance and inadequate insurance coverage are the most significant barriers to access to such care (Brown, ed., 1988, pp. 54-114). Interventions to improve the life chances of low-birth weight babies (as described by Hertzman and Wiens, 1996, p. 1089) and educational programs to improve the health related behaviours of pregnant women in certain high-risk groups (Mrazek and Haggerty, eds., 1994, p. 232) thus might have significant effects in improving health outcomes. So, for that matter, might national health insurance on the Canadian model. None of these measures would eliminate all obstacles to adequate prenatal care, or even all the obstacles unequivocally related to mothers' economic situations. Each, however, could be a necessary (although not sufficient) condition for the effectiveness of a range of other, more targeted interventions.

What does it mean to prevent mental illness? Here as elsewhere, definitions are not necessarily given, nor are they neutral in their impact on research outcomes and policy choices. We must, therefore, be methodologically self-conscious in formulating and applying them. One approach to definition adopts the familiar triad of primary, secondary and tertiary prevention: primary prevention refers to efforts to reduce a disease's rate of occurrence; secondary prevention refers to efforts to reduce prevalence, for example through more effective case finding; and tertiary prevention refers to efforts to reduce severity and adverse consequences or to prevent relapse (Eisenberg, 1992, pp. 231-232; Greenfield and Shore, 1995). An alternative view was adopted in 1994 by a committee of the U.S. Institute of Medicine (IOM) (Mrazek and Haggerty, eds., 1994), which defined prevention strictly in terms of avoiding the initial onset of a disorder. All interventions contributing to recovery and rehabilitation or reducing the probability of subsequent episodes were regarded instead as elements of treatment protocols. The success of preventive

initiatives was thereby transformed into a dichotomous variable: mental illness in any specific individual was prevented/not prevented.

This definition has the undeniable attraction of precision, but it may be a false precision. The approach of the IOM committee can very easily have the effect of shrinking the universe of potential preventive interventions, in contrast to a separate body of interventions that qualify as treatment or rehabilitation, and which are presumably well understood. Numerous interventions may fit clearly into neither category, yet may still be extremely important in affecting the course of mental illness and the associated suffering. It may be more valuable, and more consistent with public health objectives as they have been widely recognized outside the mental health field, to think in terms of a continuum from primary prevention through treatment and other clinical interventions, on which the location of various dividing lines is treated as a matter of secondary importance.

For example, for purposes of designing interventions to weaken the feedback loops³ that link economic situation and major depression, consider the frustrations and adversities experienced on a daily basis by one adult member of a household with two income earners. Each has a relatively secure job with negotiated sick leave provisions that make possible the occasional ‘mental health day’ (a wise and revealing phrase). Then compare his situation with that of a single mother juggling child care requirements with the demands of a job in retail or fast-food (cf. Ehrenreich, 1999) ... or worse, dealing with the sometimes capricious demands of a social service bureaucracy (Capponi, 1999; Edin and Lein, 1997; Funicello, 1993). Should interventions that ameliorate the adversities experienced by people in this second category be considered effective if

3. We explain our preference for the terminology of feedback loops in section IV, *infra*.

they ‘only’ improve chances of rehabilitation, facilitate adaptation or reduce the likelihood of subsequent illness-related disability?

In order to constitute prevention as defined by the IOM committee, interventions must be demonstrably associated with reduced incidence, or initial occurrence, of mental illness. The problem is complicated because economic situation may affect the risk of mental illness either directly or through a variety of intervening variables, such as community disorganization or levels of violence. Economic situation may also be a variable that mediates the effects of other demographic characteristics on the incidence and course of mental illness.⁴ Further, it may be difficult to separate income-related effects on the mental health of children and adolescents from effects on that of their parents. Indeed, it would be surprising if this were not the case.

As a general observation, by no means confined to interventions that address variables related to economic situation, prevention may emerge as possible at a variety of scales or levels, with different actors, obligations, and degrees of ethical and logistical complexity involved. An ethic of public health as applied in the mental health field might regard the question ‘is it prevention?’ as distinctly secondary to the question ‘does it help?’ Pointing out the importance of this distinction is valuable, in and of itself.

4. The Epidemiologic Catchment Area (ECA) study in the United States found that African-Americans suffered from higher historical and current incidence of mental disorder than did whites and Hispanics. “However, this difference in rates is confounded by socioeconomic status (SES). When SES is controlled, rates for African-Americans are no higher than for whites” (Regier and Kaelber, 1995, p. 143). This does not mean that race is unrelated to mental disorder; it simply means that the effects of race may be mediated by the all-too-familiar variable of race-related economic inequality. The same may well be true for gender, disability, and age. In addition, more sensitive and sophisticated indicators of economic situation than conventional measures of SES are almost certainly needed for purposes of further research.

III. Standards of Proof

Health economist Robert Evans has argued that an important “asymmetry of onus” in the evidentiary criteria applied to prevention. Many complicated and expensive treatments have come into widespread use despite the lack of conclusive evidence, specifically evidence from randomized clinical trials, that shows their benefit in terms of such outcome measures as life expectancy and quality of life. By contrast, “in preventive health care, if we do not know, we do not do it” (Evans, 1993, p. 55).

This observation does not imply criticism of clinicians, who may have several good reasons to make some treatments available even in the absence of evidence from controlled trials. Many of the available outcome measures have important limitations. Especially with respect to psychiatric medications, an important ethical debate continues about (1) when placebos may justifiably be used in controlled trials, and the ethical requirements governing the design of trials that incorporate a placebo arm, and (2) when trials must be ‘unblinded’ – in other words, when patients must be informed about the kind of treatment they are receiving and about the results of the trial so far.⁵ These are major questions that continue to demand ethical investigation. Finally, a ubiquitous question about evidence-based medicine and the application of practice guidelines is the extent to which even the best designed studies at the population level can be used, on their own, to assess the appropriateness of a clinical intervention with respect to any individual patient.

5. Critiques of placebo controlled trials of antipsychotic medications can be found in Freedman, Weijer and Glass (1996) and Freedman, Glass and Weijer (1996). For defences and replies see Addington (1995) and Carpenter, Schooler and Kane (1997).

The IOM committee demonstrated the importance of Evans' insight when it identified randomized controlled trials, ideally repeated to test the intervention's generalizability, as the evidentiary gold standard in prevention research (Mrazek and Haggerty, eds., 1994, pp. 373-376). The implications for prevention policy of setting such a high standard of proof are evident from the report's finding that:

There are data that clearly show that preventive interventions can reduce risk factors that are associated with the onset of many mental disorders. However, as yet, there is *no evidence* that preventive interventions reduce the incidence of mental disorders" (Mrazek and Haggerty, 1994, p. 298, emphasis added).

How should this conclusion be used as the basis for making decisions about prevention programs and strategies? In other words, how much evidence is enough, and what values should bear on the choice of evidentiary standards? Keeping in mind the difficulties of conducting randomized controlled trials for many kinds of interventions, should evidence of reduction in risk factors be treated as sufficient to justify preventive interventions, or should evidence of the link between reduction in risk factors and reduction in the incidence of mental illness also be required?

As in the case of results from clinical trials of new or improved therapies are involved, what are the ethical implications of waiting for more conclusive evidence? Somerville (1999) points out that this issue has recently arisen in Canada, where provincial authorities have claimed in several cases that the evidence is insufficient to justify providing reimbursement for leading-edge cancer treatments through public health insurance plans.

No scientist or social scientist can answer such questions on the basis of professional competence. Instead, they demand explicit attention to the various, potentially competing values at stake and the way they affect key choices that are made about research designs: identifying

outcome measures; determining the appropriate standard of proof (how much evidence is enough); assigning the burden of proof; and selecting an appropriate scale. On this last point, consider the fact that the smaller the scale of the intervention and the fewer the confounding variables, the more likely it is that a controlled trial will result in a demonstration of success. Conversely, the larger the scale of the intervention and the more events and situations that may affect the relation (a) between the intervention and the risk factor and (b) between the risk factor and the outcome measure, the harder it becomes to demonstrate success.

This point can be illustrated using two hypothetical examples.

First, consider an imaginary quantitative study designed to compare incremental interventions like the San Francisco mood control course with interventions to alter the depressogenic background conditions that defined the high-risk population. Such a study might involve three, rather than two groups of randomly assigned subjects, with the third group not receiving the course but being offered access to relatively more generous unemployment benefits, or a fast-track application procedure for a higher level of disability benefits.⁶

Second, imagine a prospective longitudinal study designed to test the ability of social policy interventions to prevent mental illness by altering environmental variables that now affect exposure to risk factors in early childhood. Such a study might recruit pregnant, low-income single women in the United States through intensive outreach and then assign them randomly to one of two groups. The control group would receive only the income supports available since the 1996 welfare reforms, and the uneven access to prenatal and postnatal medical care of uncertain quality that goes along with their situation. The treatment group would be offered universal

6. As noted supra (note 3), a quarter of the entire San Francisco sample were out of work because of disability. On the circuitous and demanding procedure for obtaining federal disability benefits see Gellhorn (1995).

first-dollar health care coverage, as well as a Norwegian-style package of support measures for single mothers (as outlined by Kamerman, 1995, pp. 247-251). The mental health of the children in both groups, *and of the mothers*, would then be followed through at least until the children's adolescence.

To put it mildly, the barriers confronting such studies are formidable; they involve numerous variables that are normally outside the control of researchers. It would be all but impossible to control for genetic variables in the study design, thus exposing results to criticism from evolutionary psychologists. Conversely, even such an ambitious study might *understate* the effects of economic situation on mental health -- for instance, because of its inability to control for the effects of a past history of employment discrimination, or for the effects of economic adversity on mothers' preconception health status. We are left with the disquieting possibility that choosing certain research strategies and standards of proof means the big questions about preventing mental illness probably will not be studied in ways that demonstrate the effectiveness of larger-scale, contextual interventions, and even the small questions will be asked in ways that seriously circumscribe the set of possible answers.

IV. The Biological Plausibility of Prevention

An even more far-reaching illustration of the importance of methodological self-consciousness in the choice of research designs and strategies involves the biological focus that now dominates published psychiatric research, at least in North America. That orientation arguably engenders an attitude best described as biological fatalism with respect to the prospects for preventing mental illness. The perils of biological fatalism are especially important when genetic 'explanations' are

being advanced, given the role of “the gene as cultural icon” (Nelkin and Lindee, 1995) and the popularity of evolutionary explanations for patterns of human behaviour as diverse as mate preferences, fondness for gossip and distaste for eating worms (Pinker, 1997).

At one level, to the extent that we are biological beings all our behaviour (like that of any other organism) can be described in terms of ‘gene expression’.⁷ However, like most tautologies this one is of severely limited explanatory value. It is important to recall that the most sophisticated statistical calculations often show that heredity accounts for 50 percent or less of the variance in outcomes as diverse as developing schizophrenia and volunteering for combat in Southeast Asia (Jones and Cannon, 1998; Lyons et al., 1993; McGuffin, Owen and Farmer, 1995; Tsuang and Faraone, 1994). Although the influence of experience on brain structure and function is perhaps particularly evident in childhood and adolescence, it is by no means limited to early life (Nelson and Bloom, 1997). Stressful life events and situations throughout the life course appear to play an important role in the onset of major depression (Brown, 1997; Cooper and Paykel, 1993), and probably in the severity of schizophrenic symptoms and the timing of onset (Bebbington and Kuipers, 1993; Norman and Malla, 1993).

Generalizations about environmental change and the prevention of mental illness are thoroughly unwise: mental illness is an imprecise “supercategory” (Eisenberg, 1977, p. 903) and the environmental factors that interact with an individual’s genotype may be as diverse as oxygen deprivation or inadequate nutrition during pregnancy, central nervous system infections in childhood (Jones and Cannon, 1998, p. 19) and unwanted pregnancy (Myhrman et al., 1996) – all of which are at least potentially implicated as risk factors for schizophrenia. Nevertheless, it is

7. Thus Kendler (1997, p. 7) has remarked on geneticists’ tendency “to define the environment as ‘things that get in the way of gene expression’.”

important to reject the presumption, which may or may not be stated explicitly, that causal linkages run primarily from the biological substrate to subjective or psychological experience: in other words, primarily from brain to mind. Indeed, as contemporary brain research deepens our understanding of the physiological processes that can be initiated or intensified by changes in the social environment, it undermines the viability of any dichotomy between mind and brain (Eisenberg, 1986; Eisenberg, 1995; Valenstein, 1998). When environment and genetics interact the direction of causation is often far from self-evident; and multiple feedback loops, both positive and negative, are involved (Kendler, 1995; Rutter et al., 1997). Indeed, the concept of a feedback loop is little used in mental illness research although, because of its intrinsically dynamic orientation, it may be more precise than the currently preferred vocabulary of risk and protective factors in describing the actual interaction of environmental variables in and with people's lives (Eisenberg, 1997, p. 69; Brenner et al., 1992). For purposes of research on prevention, it is important to recognize that environmental characteristics of concern may be experienced by everyone, or almost everyone, in a particular society or subgroup within a society. Thus, Jones and Cannon (1998, p. 14-15) remark that if everyone smoked, then the incidence of lung cancer would appear to be genetically determined. Analogously, the incidence of any specific mental illness will appear to be genetically determined in subgroups within a particular society that share exposure to a particular set of social risks.

The issue here is one of how the choice of an intellectual starting point affects the conclusions that are plausible, or even possible. Research that does not start by according a privileged position to the background assumptions of evolutionary biology – leading to such manifestations of genetic determinism as the claim that children and adolescents seek out, influence or even “create” their environments based on genetic predispositions (see e.g. Scarr,

1992; Scarr, 1993) – tends to generate findings that emphasize the significance of environmental factors, broadly defined, and that undermine neat distinctions between the biological and the behavioural. The example of PTSD is useful because of the dramatic illustrations it provides of the interaction between psychology and biology (Pitman, 1997; van der Kolk, 1994). For example, a variety of neuroendocrinal abnormalities as well as smaller hippocampal size, as documented by brain imaging, have been associated with PTSD (Bremner et al., 1995; Yehuda et al., 1993). Research on the relation between stress and behaviour more generally emphasizes the perils of simplistically assuming that genetics provides the only meaningful account of intergenerational transmission of illness. Stephen Suomi's work on the effects of maternal rearing practices and social environment on infant primates (Suomi, 1997a; 1997b; see also Kraemer and Clarke, 1996) suggests that

... effects of early experiences are not limited to behavioural phenomena but instead can encompass a wide range of biological functioning. Indeed, research with monkeys has shown that even those behavioural and physiological processes that have highly heritable features can be substantially modified by certain early experiences (Suomi, 1997a, p. 181).

Suomi hypothesized three distinct *nongenetic* pathways for the intergenerational transmission of the effects of traumatic stress (Suomi and Levine, 1998). Fleming, O'Day and Kraemer (1999) have described at the molecular level the various mechanisms by which such transmission might occur, and Francis et al. (1999) have identified demonstrated the operation of one such mechanism across generations of rats. The implications for the etiology of mental illness in human populations are, to say the least, provocative when we consider the evidence for familial transmission of such effects.

Apart from the specific case of PTSD, heredity's contribution to the risk for such conditions as major depression and schizophrenia is well established, as is the substantial genetic contribution to variability in individual responses to stress (Eley and Plomin, 1997; Kendler et al., 1995). However, the etiological significance of stressful events and situations may be underestimated in research designs that restrict their consideration to discrete events occurring over a limited period of time, often less than a year, or fail to take into account broader situational factors. For example, one British study found that "significant involvement in domestic roles" by men in a household apparently acted as a protective factor against depression when a shared crisis involving "children, housing and reproduction" confronted both members of a couple. When the household was characterized by a pronounced gender-based division of labour, on the other hand, women were five times as likely as men to experience a depressive episode (Nazroo, Edwards and Brown, 1997). Adversities that are related to economic situation constitute another broad category of such situational factors. It could be argued that such a longer-range perspective invites confusion about causation. In other words, do disorders like schizophrenia or depression result in a 'downward drift' into life situations where adversities are more frequent? They almost certainly do, although the relative plausibility of downward drift and an alternative explanation, in which the environmental factors associated with social and economic disadvantage result in increased incidence of mental illness, is likely to vary not only with the disorder in question (Dohrenwend et al., 1992) but also, perhaps, with the study population.

V. The Ethics and Economics of Social Risk Reduction

Here we return to an issue identified at the start of the article. Even when the effectiveness of preventive interventions can be demonstrated, much of the contemporary literature suggests that the desirability of undertaking them is not self-evident. The IOM report refers to cost-effectiveness or benefit-cost ratio as a criterion that should be incorporated into the design of research on preventive interventions (Mrazek and Haggerty, eds., 1994, pp. 240, 285, 405-409). A Canadian study of the economics of preventing mental illness among children cites “a need to find the optimal combination of approaches to reduce the burden of suffering that is affordable since the costs of interventions can be enormous, yet are balanced by the cost of the illness itself” (Offord et al., 1997, p. 3, see also pp. 14-16). Such approaches are typical of the “social investment model” (Hertzman and Wiens, 1996, p. 1092) adopted by many advocates of preventive interventions, yet they are a long way from the broad vision of public health identified earlier in this article.

It is probably true that “investing in high quality social and physical environments makes good economic sense” (Guy, ed., 1997, p. 21), at least some of the time. Perhaps more importantly, any credible evidence to that effect gives advocates of preventive intervention the tactical advantage of arguing from enlightened self-interest, at least at the societal level. This is likely to be more effective than invoking considerations of beneficence or protecting the vulnerable, since such themes seem to be increasingly peripheral to the main concerns of government. Many health economists concede the malleability of economic analysis with respect to such issues as the definition of relevant costs, the choice of discount rates, and the difficulty of valuing suffering, disability, and reductions in longevity. Indeed, for purposes of economic analysis no value at all may be attached to the reduction of suffering *per se*. Unfortunately, it is not clear how thoroughly

these uncertainties are understood by the users of the resulting numbers. For purposes of ethical analysis it is therefore critically important to ask under what conditions, and for what reasons, the relevant decision-makers would be justified in refusing to support programs or interventions on the basis that they will not pay for themselves. One can readily think of situations in which defining the rationale for preventive interventions in economic terms is intuitively troubling⁸ and we normally recoil from such forms of triage, at least when they are presented explicitly, for a variety of reasons. They imply the possibility, and perhaps the inevitability, of age discrimination (Avorn, 1984; Somerville, 1986), as well as the possibility that those already vulnerable for one of many reasons will be further disadvantaged by moving to the end of the queue for resources that would enable them to minimize their suffering and to cope as best they can with a variety of reduced capacities. For instance, consider a hypothetical situation in which (a) a randomized controlled trial demonstrates the effectiveness of a package of measures to reduce the subsequent incidence of mental illness and other adverse outcomes among severely disadvantaged children, but (b) a cost-benefit analysis shows a higher cost-benefit ratio, as determined with reference to subsequent outcomes, from spending the money that would finance those measures on further improving the situation of middle class children who are already relatively comfortable? Many conceptions of distributive justice fail to provide a convincing rationale for allocations of resources that effectively increase inequalities of situation or opportunity. By the same token informed consent considerations, which in the context of psychiatric genetics have appropriately been called “the cornerstone of ethical research and practice,” argue against a variety of public health research designs and practices that leave the most disadvantaged worse off. (Faraone, Tsuang and Tsuang, 1999, p. 231)

8. On the importance of “intuitive dissatisfaction” as an ethical resource see Nagel (1991, p. 7).

It must also be acknowledged that research is carried out within a social and economic context, and research directions may be shaped by economic or professional interests. Valenstein (1998) emphasizes this point in his discussion of the connections between the profitability of pharmacotherapy and the ascendancy of biological psychiatry. More generally, as universities, granting agencies and private sources of research funding alike come to organize their priorities around the possibilities for commercialization, prevention research is likely to be short-changed simply because it is difficult, if not impossible, to envision a scenario in which intellectual property rights in findings could be asserted in a way comparable to the routine filing of patent applications for pharmaceutical compounds or human genetic material. Conversely, when research on interventions with a potential for social risk reduction might strengthen the case for a new or additional commitment of resources, institutional responses are likely to resemble those encountered in the case of very expensive cancer treatments (Somerville, 1999). Kim Hopper (1996, p. 205), whose particular concern is the tension between historical conceptions of public health and the contemporary inadequacy of services for the homeless mentally ill, warns of:

... the prospect that the public fisc could substitute for the common weal as the reference point for 'harm,' such that 'wasteful' or 'excess' spending will be seen as the functional equivalent of assault on the body politic.

As disturbing as this prospect may be, even more disturbing is the possibility that the values of fiscal conservatism could be cloaked in the superficially unexceptionable guise of scientific conservatism. Thus, as in the example of cancer treatments, demanding (perhaps impossibly demanding) standards of proof may be adopted as preconditions for institutional action, because they increase the credibility of claims that 'no benefit has been demonstrated' or, simply, that 'nothing works'. The economies achieved by asserting such claims may be consistent with

organizational objectives, whether they involve raising share prices or lowering public expenditures, but such objectives cannot in themselves claim any distinctive ethical status.

VI. Conclusion

In keeping with our transdisciplinary orientation, in this article we have tried to do several things at once. We have done so because of the urgent need for those involved with research on mental illness, in any capacity, to look beyond their own specific professional competence or institutional responsibility. Specifically, ethical inquiry in this area needs to expand beyond its present intellectual boundaries if it is to fulfil its proper function of embedding ethics in our approaches to mental illnesses and people suffering from them. We have identified a number of key methodological value judgments in contemporary research on preventing mental illness, focusing on the possibilities for its prevention, in order to demonstrate the need for methodological self-consciousness on the part of researchers who may be too ready to see both their choice of research designs and the findings that result as value-free. And we have suggested that efforts to prevent mental illness must draw on approaches from other areas of public health practice, where the idea of social risk reduction is more widely accepted even if not described in those words. In mental health as elsewhere, we are on the brink of developing a far richer and more paradigm for both conducting and applying research. It will take courage and humility to pursue this paradigm, and to put it into practice.

Acknowledgments

An earlier version of this article was presented at the XXIVth International Congress on Law and Mental Health, Toronto, June 1999. All views expressed are exclusively those of the authors.

References

- Addington, D. (1995) The Use of Placebos in Clinical Trials for Acute Schizophrenia. *Canadian Journal of Psychiatry* 40, 171-176.
- Albee, G. (1996). Revolutions and Counterrevolutions in Prevention. *American Psychologist* 51, 1130-1133.
- Avorn, J. (1984). Benefit and Cost Analysis in Geriatric Care: Turning Age Discrimination Into Health Policy. *New England Journal of Medicine* 310, 1294-1301.
- Beauchamp, T. Childress, J. (1994). *Principles of Biomedical Ethics*, 4th ed. Oxford University Press, New York.
- Bebbington, P. and Kuipers, L. (1993). Social Causation of Schizophrenia. In *Principles of Social Psychiatry*, ed. D. Bhugra and J. Leff, pp. 82-98. Blackwell, Oxford.
- Bowman, M. (1999). Individual Differences in Posttraumatic Distress; Problems with the DSM-IV Model. *Canadian Journal of Psychiatry* 44: 21-33.
- Breakey, W. (1996). Prevention. In *Community Mental Health Services*, ed. W. Breakey, pp. 323-336. Oxford University Press, New York.
- Bremner, J., Randall, P., Scott, T., Bronen, R., Seibyl, J., Southwick, S., Delaney, R., McCarthy, G., Charney, D., Innis, R. (1995). MRI Based Measurement of Hippocampal Volume in Patients with Combat-Related PTSD. *American Journal of Psychiatry* 152, 973-981
- Brenner, H., Hodel, B., Genner, R., Roder, R. and Corrigan, P. (1992). Biological and Cognitive Vulnerability Factors in Schizophrenia: Implications for Treatment. *British Journal of Psychiatry* 161, suppl. 18, 154-163.
- Brown, G. (1997). A Psychosocial Perspective on the Aetiology of Depression. In *Depression: Neurobiological, Psychopathological and Therapeutic Advances*, ed. A. Honig and H.M. van Praag, pp. 343-362. John Wiley, Chichester.
- Brown, S., ed. (1998). *Prenatal Care: Reaching Mothers, Reaching Infants*, Committee to Study Outreach for Prenatal Care, Institute of Medicine. National Academy Press, Washington, D.C.
- Capponi, P. (1999) *The War at Home: An Intimate Portrait of Canada's Poor*. Viking, Toronto.
- Carpenter, W., Schooler, N. and Kane, J. (1997) The Rationale and Ethics of Medication-Free Research in Schizophrenia. *Archives of General Psychiatry* 54, 401-07.

- Cohen, C. (1993). Poverty and the Course of Schizophrenia: Implications for Research and Policy. *Hospital and Community Psychiatry* 44, 951-958.
- Cooper, Z., Paykel, E. (1993). Social Factors in the Onset and Maintenance of Depression. In *Principles of Social Psychiatry*, ed. D. Bhugra and J. Leff, pp. 99-121. Blackwell, Oxford.
- Coughlin, S. and Beauchamp, T., eds. (1996) *Ethics and Epidemiology*. Oxford University Press, New York.
- Dohrenwend, B., Levav, I., Shrout, P., Schwartz, S., Naveh, G., Link, B., Skodol, A., Stueve, A. (1992). Socioeconomic Status and Psychiatric Disorders: The Causation-Selection Issue. *Science* 255, 946-952.
- Edin, K., Lein, L. (1997). *Making Ends Meet: How Single Mothers Survive Welfare and Low-Wage Work*. Russell Sage Foundation, New York.
- Ehrenreich, B. (1999). Nickel-and-Dimed: On (not) Getting By in America. *Harper's Magazine*, January, 37-52.
- Eisenberg, L. (1986). Mindlessness and Brainlessness in Psychiatry. *British Journal of Psychiatry* 148, 499-508.
- Eisenberg, L. (1992). Child Mental Health in the Americas: A Public Health Approach. *Bulletin of the Pan-American health Organization* 26, 230-241.
- Eisenberg, L. (1995). The Social Construction of the Human Brain. *American Journal of Psychiatry* 152, 1563-1575.
- Eisenberg, L. (1997). Psychiatry and Health in Low-Income Populations. *Comprehensive Psychiatry* 38, 69-73.
- Eley, T., Plomin, R. 1997. Genetic Analyses of Emotionality. *Current Opinion in Neurobiology* 7, 279-284.
- Evans, R. (1993). Ethical Ambiguities and Economic Consequences in the Allocation of Health Care. In *Health Care, Ethics and Law*, ed. B. Dickens and M. Ouellette, pp. 46-58. Les Éditions Thémis, Montréal.
- Evans, R., Stoddart, G. (1990). Producing Health, Consuming Health Care. *Social Science and Medicine* 31, 1347-1363.
- Faraone, S.V. Tsuang, M.T. Tsuang, D.W. (1999). *Genetics of Mental Disorders: A Guide for Students, Clinicians, and Researchers*. Guilford Press, New York.

- Farley, M. Barkan, H. (1998). Prostitution, Violence, and Posttraumatic Stress Disorder. *Women & Health* 27, 37-49.
- Farley, M. Baral, I. Kiremire, M. and Sezgin, U. (1998). Prostitution in Five Countries: Violence and Post-Traumatic Stress Disorder. *Feminism & Psychology* 8, 405-426.
- Fleming, A.S., O'Day, D.H. and Kraemer, G.W. (1999). Neurobiology of Mother-Infant Interactions: Experience and Nervous System Plasticity Across Development and Generations. *Neuroscience and Biobehavioral Reviews* 23, 673-685.
- Francis, D., Diorio, J., Liu D., Meaney M.J. (1999). Nongenomic Transmission Across Generations of Maternal Behavior and Stress Responses in the Rat. *Science* 286, 1155-1158.
- Freedman, B., Weijer C. and Glass, K. (1996). Placebo Orthodoxy in Clinical Research I: Empirical and Methodological Myths. *Journal of Law, Medicine & Ethics* 24, 243-51.
- Freedman, B., Glass, K. and Weijer, C. (1996) Placebo Orthodoxy in Clinical Research II: Ethical, Legal and Regulatory Myths. *Journal of Law, Medicine & Ethics* 24, 252-59.
- Funiciello, T. (1993). *Tyranny of Kindness*. Atlantic Monthly Press, New York.
- Gellhorn, G. (1995). Disability and Welfare Reform. *Fordham Urban Law Journal* 22, 961-1008.
- Greenfield, S., Shore, M. (1995). Prevention of Psychiatric Disorders. *Harvard Review of Psychiatry* 3, 115-129.
- Guy, K. ed. (1997) *Our Promise to Children*. Canadian Institute of Child Health, Ottawa.
- Harrison, G., Glazebrook, C., Brewin, J., Cantwell, C., Dalin, T., Fox, R., Jones, P. and Medley, I. (1997). Increased Incidence of Psychotic Disorders in Migrants from the Caribbean to the United Kingdom. *Psychological Medicine* 27, 799-806.
- Hartmann, L. (1992). Presidential Address: Reflections on Humane Values and Biopsychosocial Integration. *American Journal of Psychiatry* 149, 1134-1141.
- Heller, K. (1996). Coming of Age of Prevention Science. *American Psychologist* 51, 1123-1127.
- Hertzman, C. and Wiens, M. (1996). Child Development and Long-term Outcomes: A Population Health Perspective and Summary of Successful Interventions. *Social Science and Medicine* 43, 1083-1095.
- Hiday, V. (1997). Understanding the Connection Between Mental Illness and Violence. *International Journal of Law and Psychiatry* 20, 399-417.

Hopper, K. (1996). Regulation from Without: The Shadow Side of Coercion. In *Coercion and Aggressive Community Treatment*, ed. D. Dennis and J. Monahan, pp. 197-212. Plenum, New York.

Kamerman, S. (1995). Gender Role and Family Structure Changes in the Advanced Industrialized West. In *Poverty, Inequality and the Future of Social Policy*, ed. K. McFate et al., pp. 231-256. Russell Sage Foundation, New York.

Kendler, K. (1995). Genetic Epidemiology in Psychiatry: Taking Both Genes and Environment Seriously. *Archives of General Psychiatry* 52, 895-899.

Kendler, K. (1997). The Genetic Epidemiology of Psychiatric Disorders: A Current Perspective. *Social Psychiatry and Psychiatric Epidemiology* 32, 5-11.

Kendler, K., Kessler, R., Walters, E., MacLean, C., Sham, P., Neale, M., Heath, A. and Eaves, J. (1995). Stressful Life Events, Genetic Liability and Onset of an Episode of Major Depression in Women. *American Journal of Psychiatry* 152, 833-842.

Kraemer, G. and Clarke, S. (1996). Social Attachment, Brain Function, and Aggression. *Annals of the New York Academy of Sciences* 794, 121-135.

Krieger, N. and Zierler, S. (1996). What Explains the Public's Health? A Call for Epidemiologic Theory. *Epidemiology* 7, 107-109

Kuhn, T. (1970). *The Structure of Scientific Revolutions*, 2nd edition. University of Chicago Press, Chicago.

Longino, H. (1990). *Science as Social Knowledge: Values and Objectivity in Scientific Inquiry*. Princeton University Press, Princeton, NJ.

Lyons, M., Goldberg, J., Eisen S., True, W., Tsuang, M., Meyer, J. and Henderson, W. (1993). Do Genes Influence Exposure to Trauma? A Twin Study of Combat. *American Journal of Medical Genetics* 48, 22-27.

Mann, J. (1997). Medicine and Public Health, Ethics and Human Rights. *Hastings Center Report* 27, no. 3, May-June, 6-13.

McGuffin, P., Owen, M. and Farmer, A. (1995). Genetic Basis of Schizophrenia. *The Lancet* 346, 678-682.

McMichael, A. (1995). The Health of Persons, Populations and Planets: Epidemiology Comes Full Circle. *Epidemiology* 6, 633-636.

Mrazek, P. and Haggerty, R., eds. (1994). *Reducing Risks for Mental Disorders: Frontiers for Preventive Intervention Research*, Committee on Prevention of Mental Disorders, Institute of Medicine. National Academy Press, Washington, D.C.

Muñoz, R., Ying, Y., Bernal, G., Pérez-Stable, E., Sorensen, J., Hargreaves, W., Miranda, J. and Miller, L. (1995). Prevention of Depression with Primary Care Patients: A Randomized Controlled Trial. *American Journal of Community Psychology* 23, 199-222.

Myhrman, A., Rantakallio, P., Isohanni, M., Jones, P., Partanen, U. (1996). Unwantedness of a Pregnancy and Schizophrenia in the Child. *British Journal of Psychiatry* 169, 537-540.

Nagel, T. (1991). *Equality and Partiality*. Oxford University Press, New York.

Nazroo, J., Edwards, A. and Brown, G. (1997). Gender Differences in the Onset of Depression following a Shared Life Event: A Study of Couples. *Psychological Medicine* 27, 9-19.

Nelkin D.M., Lindee S. (1995.) *The DNA Mystique: The Gene as a Cultural Icon*. W.H. Freeman, New York.

Nelson, C.A., and Bloom, F.E. (1997). Child Development and Neuroscience. *Child Development* 68, 970-987.

Norman, R. and Malla, A. (1993). Stressful Life Events and Schizophrenia I: A Review of the Research. *British Journal of Psychiatry* 162, 161-166.

Offord, D., Kraemer, H., Kazdin, A., Jensen, P. and Harrington, R. (1997). *Lowering the Burden of Suffering from Child Psychiatric Disorder: Trade-Offs Among Clinical, Targeted and Universal Interventions*. Program in Human Development Working Paper No. 30. Canadian Institute for Advanced Research, Toronto.

Paris, J. (1999). Does Stress Cause Posttraumatic Stress Disorder? (Editorial). *Canadian Journal of Psychiatry* 44, 20.

Pearce, N. (1996). Traditional Epidemiology, Modern Epidemiology, and Public Health. *American Journal of Public Health* 86, 678-683.

Pinker, S. (1997). *How the Mind Works*. W.W. Norton, New York.

Pitman, R. (1997). Overview of Biological Themes in PTSD. *Annals of the New York Academy of Sciences* 821, 1-9

Regier, D. and Kaelber, C. 1995. The Epidemiologic Catchment Area (ECA) Program: Studying the Prevalence and Incidence of Psychopathology. In *Textbook in Psychiatric Epidemiology*, ed. M. Tsuang, M. Tohen and G. Zahner, pp. 135-155. John Wiley, New York.

Reiss, D., Plomin, R. and Hetherington, M. (1991). Genetics and Psychiatry: An Unheralded Window on the Environment. *American Journal of Psychiatry* 148, 283-291.

Remington, Richard D. et al. (1988) *The Future of Public Health*. Committee on the Future of Public Health, Institute of Medicine. Washington, D.C.: National Academy Press.

Rutter, M., Dunn, J., Plomin, R., Simonoff, E., Pickles, A., Maughan, B., Ormel, J., Meyer, J. and Eaves, L. (1997). Integrating Nature and Nurture: Implications of Person-Environment Correlations and Interactions for Developmental Psychopathology. *Development and Psychopathology* 9, 335-364.

Scarr, S. (1992). Developmental Theories for the 1990s: Development and Individual Differences. *Child Development* 63, 1-19.

Scarr, S. (1993). Biological and Cultural Diversity: The Legacy of Darwin for Development. *Child Development* 64, 1333-1353.

Somerville, M. (1986). Should the Grandparents Die? Allocation of Medical Resources with an Aging Population. *Law, Medicine and Health Care* 14, 158-163.

Somerville, M. (1989). Weaving 'Birth' Technology into the 'Value and Policy Web' of Medicine, Ethics and Law: Should Policies on 'Conception' be Consistent? *Nova Law Review* 13, 515-608.

Somerville, M. (1993). The Song of Death: The Lyrics of Euthanasia. *Journal of Contemporary Health Law and Policy* 9, 1-76.

Somerville, M. (1999). The Ethics and Law of Access to New Cancer Treatments. *Current Oncology* 6, 161-174.

Suomi, S. (1997a). Early Determinants of Behaviour: Evidence from Primate Studies. *British Medical Bulletin* 53, 170-184.

Suomi, S. (1997b). Long-term Effects of Different Early Rearing Experiences on Social, Emotional, and Physiological Development in Nonhuman Primates. In *Neurodevelopment and Adult Psychopathology*, ed. M. Keshavan and R. Murray, pp. 104-116. Cambridge University Press, Cambridge.

Suomi, S. and Levine, S. (1998). Psychobiology of Intergenerational Effects of Trauma: Effects from Animal Studies. In *International Handbook of Multigenerational Legacies of Trauma*, ed. Y. Danieli, pp. 605-619. Plenum, New York.

Tsuang, M. and Faraone, S. (1994). The Genetic Epidemiology of Schizophrenia. *Comprehensive Therapy* 20, 130-135.

Valenstein, E. (1998) *Blaming the Brain: The Truth About Psychiatric Drugs*. Free Press, New York.

van der Kolk, B. (1994). The Body Keeps the Score: Memory and the Evolving Psychobiology of Posttraumatic Stress. *Harvard Review of Psychiatry* 1, 253-265.

Ware, N. and Goldfinger, S. (1997). Poverty and Rehabilitation in Severe Psychiatric Disorders. *Psychiatric Rehabilitation Journal* 21, 3-9.

Yehuda, R., Giller, E., Southwick, S., Lowy, M., Mason, J. (1993). Hypothalamic Pituitary Adrenal Dysfunction in Posttraumatic Stress Disorder. *Biological Psychiatry* 30, 1031-1048.